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## CLINICAL CASE

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### SUBACUTE THYROIDITIS CAN BE CONSIDERED A COMPLICATION OF SARS-COV-2 INFECTION-CASE PRESENTATION

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#### ABSTRACT

*The Sars Cov-2 virus produced an impact on the health system never seen before in modern times. Due to the large number of patients infected, clinical practitioners but also pathologists started to observe that other organs besides the lungs were damaged by the virus. The mechanisms through which this happens are not entirely clear mainly due to the fact that this is a novel disease and we are still learning new information on a daily basis. This multi-visceral approach of the virus to the human body wreaks havoc and is hard to contain. Our study and case presentation focused on the impact the Sars Cov-2 virus has on the thyroid gland, clinical signs, and treatment options.*

**KEYWORDS:** COVID-19, thyroid, SARS-CoV-2, De Quervain, thyroiditis

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#### INTRODUCTION

The Covid-19 pandemic has emerged with incredible consequences both economically and in terms of the loss of human life for the entire world. As the study of the virus has progressed new ways and organs that are affected besides the respiratory system have been identified. One of these is the endocrine system and in particular, the thyroid gland, and going more in-depth it has been observed that the Covid-19 virus can generate subacute thyroiditis demonstrated by multiple recent cases reports [1-4]. Subacute thyroiditis or De Quervain's thyroiditis is defined as a pathology in which one of the main clinical manifestations is a pain in the cervical region accompanied by signs and

symptoms of thyrotoxicosis. Although it has multiple etiologies one of the known and demonstrated causes is a viral infection and the case of Covid-19 is no different [5].

Taking into account that the virus infected more than 578 million worldwide as reported by worldmeter.com accessed on 05.08.2022 and possibly many more who were not diagnosed. We can deduct that even if a small percentage of the infected patients have thyroid implications this translates into large absolute numbers thus making this condition important and the physician needs to be made aware of it.

The reported signs and symptoms of subacute thyroiditis include neck pain during movement especially in the anterior region which

can migrate to the posterior region, local edema, and tenderness [6].

High blood values of FT4 can be confirmed, also as demonstrated in our patient at ultrasonography one can observe a heterogeneous gland with low blood supply. Low scintigraphic fixation has also been reported [6].

Our study aims to bring into the spotlight the fact that the thyroid gland can be exposed to the SARS-Cov2 virus, and that these patients have specific clinical manifestations thus it needs to be recognized by the clinician. Rapid and correct treatment leads to the complete remission of symptoms.

### CASE PRESENTATION

We present the case of a 45 years old male who was infected with the SarsCov-2 virus 6 weeks prior. At presentation, a negative PCR test was confirmed with negative IgM antibodies and positive IgG antibodies. The patient complained of fever (39C), night sweats, diffuse muscle pain, extreme fatigue, anterior cervical pain, loss of appetite, insomnia, and weight loss (4kg 7 days). The clinical evaluation confirmed high blood pressure of 160/100 mmHg and tachycardia of 109 BPM. The intense pain was noted on palpation in the anterior cervical region.

The paraclinical investigations identified anemia with Hb levels of 11g/dL, inflammatory syndrome with VSH>140 mm/h, Fibrinogen 698 mg/dL, and Protein C values of 137.43 mg/l. Also, to be noted the patient had negative A-TPO antibodies, low TSH < 0.00 mU/L, and increased FT3 >8.52mol/L and T4>3.94 values. The rest of the blood work was within normal parameters. Thyroid ultrasonography was done which confirmed the following (Figure 1, Figure 2).

The patient was started on Prednison 5mg/ 8 capsules/ day for 7 days followed by 4 capsules - for 7 days, followed by 2 capsules - for 7 days followed by 1 capsule for 3 days. Also, Propanolol was added to 40mg in doses of ¼ capsule 4x/day. The patient was re-evaluated after six weeks. The clinical symptoms receded with FT3, FT4, TSH, and inflammatory markers in normal ranges.

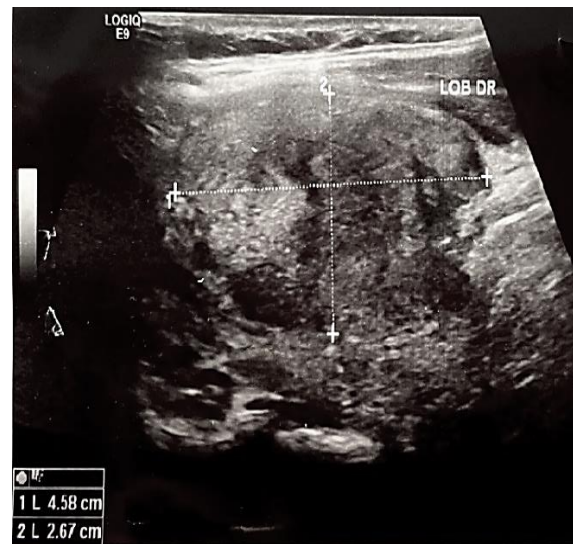


Figure 1 – Right thyroid lobe



Figure 2 – Left thyroid lobe

### DISCUSSIONS

The SARS-COV-2 virus mainly affects the pneumocytes. The structure that the virus recognizes for the “lock-key” mechanism is ACE2-protein or the angiotensin-converting enzyme 2. But this protein which is used as a travel mechanism is expressed by other organs, especially the endocrine system such as the pituitary gland, pancreas, ovary, thyroid, or adrenal glands so as the blood viremia reaches high enough values the virus could also theoretically lock on to these receptors [8]. One of the mechanisms which lead to this complication in the case of viral infection is molecular mimic by which T-cells are stimulated due to the increased antibody production and through the cytolytic effect they identify and destroy thyroid cells as they are wrongly

recognized as non-self [8]. It is a well-known fact that an infection with a virus can trigger an autoimmune reaction from the host organism that can act as the trigger factor for a lifetime disease such as for example post-varicella myasthenia gravis disease [9].

Vojdani et al have observed that the antibodies produced by the B cells which should lock on the spike antigen of the SARS-CoV-2 virus also interact with a specific thyroid enzyme [10]. This enzyme is called thyroid peroxidase (TPO) and by coupling to it the antibodies it generates an autoimmune response against the gland. This same mechanism happens in Hashimoto's disease. This is due to molecular mimicry, TPO mimics resembles the structure of the spike-protein [10].

This enzyme can be found in the thyroid follicles; thus, it has been demonstrated on different autopsies of patients with Covid-19 disease that there was extensive destruction of the follicular but also parafollicular cells [11]. As such, patients with thyroid disease are advised to take great caution so as not to contract the virus [11]. The clinical symptoms mimic those of subacute thyroiditis with hypo or hyperthyroidism depending on the context. These were also observed in our patients: joint and muscle pain, inflammation of the anterior cervical region, heat intolerance, tremor, fever, loss of appetite, weight loss, or weight gain [12]. If a patient presents with these symptoms who recently contracted the Sars-Cov 2 virus the clinician should investigate the thyroid disease. A trigger clinical sign should be anterior cervical swelling or pain.

The discussion in the literature provides some insight into the mechanisms through which the thyroid gland can suffer various forms of disruption in relation to Covid-19 but they do not offer a clear pathway. As such the treatment is empiric and follows principles adopted in the past for similar diseases such as immunosuppression with high doses of corticoids and symptomatics. Fortunately, these treatments offer good results as was the case with our patient but long term is required to see if this inflammatory autoimmune response leaves the patients with chronic hypothyroidism.

## CONCLUSIONS

In conclusion, the endocrine implications of the Sars-Cov2 virus are in the process of evaluation and remain currently unexplored. The reported data is scarce and based mostly on case reports. With these aspects in mind, subacute thyroiditis needs to be suspected in patients with acute cervical pain after infection with Covid-19. An endocrinologist needs to be aware of this complication and treatment.

## REFERENCES

- [1] RM Ruggeri, A Campenni, M Siracusa, G Frazzetto, D Gullo. Subacute thyroiditis in a patient infected with SARS-COV-2: an endocrine complication linked to the COVID-19 pandemic. *Hormones (Athens)*. 2020;16:1-3.
- [2] S Ippolito, F Dentali, ML Tanda. SARS-CoV-2: a potential trigger for subacute thyroiditis? Insights from a case report. *J Endocrinol Invest*. 2020; 12:1-2.
- [3] R Ruano, et al. Subacute thyroiditis might be a complication triggered by SARS-CoV-2. *Endocrinol Diabetes Nutr*. 2021; 68(10): 755–756.
- [4] A Brancatella, D Ricci, N Viola, D Sgrò, F Santini, F Latrofa. Subacute thyroiditis after SARS-COV-2 infection. *J Clin Endocrinol Metab*. 2020; 105(7):276.
- [5] R Desailoud, D Hober. Viruses and thyroiditis: an update. *Virology*. 2009; 6:5
- [6] A Tabassom, MA Edens. De Quervain Thyroiditis. [Updated 2020 Jul 15]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK526066/>.
- [7] D Dworakowska, AB Grossman. Thyroid disease in the time of COVID-19. *Endocrine*. 2020; 68:471-474.
- [8] Le Chang, Ying Yan, Lunan Wang, Coronavirus Disease 2019: Coronaviruses and Blood Safety, *Transfusion Medicine Reviews*, 2020, 34:75-80.
- [9] A Saha, P Batra, KY Vilhekar, P Chaturvedi. Post-varicella myasthenia gravis. *Singapore Med J*. 2007; 48(6):177-80.
- [10] A Vojdani, D Kharrazian. Potential antigenic cross-reactivity between SARS-CoV-2 and human tissue with a possible link to an increase in autoimmune diseases. *Clin Immunol*. 2020; 217: 108480.
- [11] L Wei, S Sun, C Xu, J Zhang, Y Xu, H Zhu et al Pathology of the thyroid in severe acute respiratory syndrome. *Hum Pathol*, 2007; 38:95–102.
- [12] A Bindra, GD Braunstein. Thyroiditis. *Am Fam Physician*. 2006;73(10):1769-6